UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF NEW YORK

MICHELE BAKER; CHARLES CARR; ANGELA CORBETT; PAMELA FORREST; MICHAEL HICKEY, individually and as parent and natural guardian of O.H., infant; KATHLEEN MAIN-LINGENER; KRISTIN MILLER, as parent and natural guardian of K.M., infant; JENNIFER PLOUFFE; SILVIA POTTER, individually and as parent and natural guardian of C.P, infant; and DANIEL SCHUTTIG, individually and on behalf of all others similarly situated,

Plaintiffs,

CIV. No. 1:16-CV-917 (LEK/DJS)

v.

SAINT-GOBAIN PERFORMANCE PLASTICS CORP., HONEYWELL INTERNATIONAL INC. f/k/a ALLIED-SIGNAL INC. and/or ALLIEDSIGNAL LAMINATE SYSTEMS, INC., E.I. DUPONT DE NEMOURS AND COMPANY and 3M CO.,

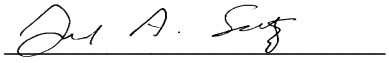
Defendants.

DECLARATION OF DAVID A. SAVITZ, Ph.D.

I, David A. Savitz, declare and state as follows:

1. I prepared the Expert Report attached as Exhibit A to this Declaration.
2. Each of the opinions in the Expert Report is stated to a reasonable degree of scientific certainty and was arrived at using reliable and generally accepted scientific methods.
3. If called as a witness, I will testify competently to the matters stated in this Expert Report.
4. I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct.

Dated: March 14, 2020



DAVID A. SAVITZ, Ph.D.

EXHIBIT A

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF NEW YORK

IN RE HOOSICK FALLS PFOA CASES **Case No. 1:19-mc-00018-LEK-DJS**

EXPERT REPORT OF DAVID A. SAVITZ, PH.D.

I. PRIOR TESTIMONY AND COMPENSATION

I have not testified as an expert at a trial or deposition in the past four years. My rate of compensation is $400 per hour.

II. QUALIFICATIONS & RELEVANT EXPERIENCE

I am Professor of Epidemiology at the School of Public Health and Professor of Obstetrics and Gynecology and Pediatrics at the Warren Alpert Medical School of Brown University in Providence, RI. A copy of my C.V. is attached as Exhibit A. I received my Ph.D. in epidemiology from the University of Pittsburgh's Graduate School of Public Health in 1982 and have had academic appointments at several academic institutions since 1981. Throughout my career, I have served as the Principal Investigator on approximately 40 public health studies and have been an author on more than 350 articles in the peer-reviewed, scientific literature and more than 20 book chapters.

I have extensive experience analyzing the health effects of PFOA in humans. I have published thirteen scientific papers in the peer-reviewed literature regarding PFOA health effects, most focused on health effects related to pregnancy and children, which are listed and highlighted in Exhibit A. I was asked to serve as a Peer Reviewer of the June 2018 Draft Toxicological Profile for Perfluoroalkyls (a class of chemicals that includes PFOA) by the United States Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR).[1] I recently chaired a scientific panel to advise the State of Michigan Science Advisory Panel on addressing the health and environmental concerns related to perfluoroalkyl substances (PFAS) exposure and provided a report entitled "Scientific Evidence and Recommendations for Managing PFAS Contamination in Michigan [2] I was also one of three epidemiologists chosen to serve on the

[1] https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf
[2] https://www.michigan.gov/documents/pfasresponse/Science_Advisory_Board_Report_641294_7.pdf.

C8 Science Panel to evaluate the probable causal link between exposure to PFOA and the development of certain diseases.

I provide this report in support of plaintiffs' Motion for Class Certification.

III. BACKGROUND ON EPIDEMIOLOGY

Epidemiology is the study of the patterns and determinants of disease in human populations, seeking an understanding of the causes of disease in order to determine needed actions to improve the health of the public. As trained epidemiologists, we conduct and review studies of populations first to determine whether there is evidence indicative of a statistical association between some potentially causative agent and a human illness or condition. This typically requires comparing the frequency of disease in a group that has relatively elevated exposure to the frequency of disease in a group that is unexposed or has a lower level of exposure.

When we determine those who are more highly exposed have an elevated risk of disease relative to those who are not, we conduct analyses needed to make an informed judgment regarding whether it is likely that the exposure has in fact caused an elevated risk of disease. While this cannot be proven with 100% certainty, the field of epidemiology has developed clear principles and methodologic tools to make a reasoned, scientifically grounded judgment. By considering alternative explanations of the association, including biases and random error, and conducting analyses to address those alternative explanations, the case for a causal interpretation can be strengthened or weakened, depending on what is found. I co-authored a book devoted to practical strategies for making such inferences in a methodical, transparent, informative manner. (Savitz DA, Wellenius GA, Interpreting Epidemiologic Evidence: Connecting Research to Applications. New York: Oxford University Press, 2016.)

The question of causality is central to epidemiology since the study of statistical associations alone without evaluating the causal significance offers no guidance for methods of preventing disease to improve public health. There is a continuum of evidence that can support causal inferences, with the example of smoking and lung cancer being one for which the evidence of a causal effect is compelling yet for many years was challenged with the simplistic mantra "correlation is not causation." The judgment to be made is whether the evidence of an association is or is not likely to reflect a causal impact. While scientific certainty of causality is difficult to establish with any exposure and may take decades of study to reach this level, epidemiologists are able to make informed use of available data to address questions of causality. By considering the body of scientific evidence and interpreting it with an appreciation of the underlying methodologic strengths and limitations, reliable judgments can be made, including when a causal link is more likely than not to be present.

An important point that needs to be emphasized is that in epidemiology, a negative study, i.e., a study that does not show a statistically significant association between an exposure and a specific illness, also needs to be scrutinized for its validity in suggesting there is no association. Just as for a positive indication of an association, studies that generate an absence of association are subject to biases and random error that can generate a false negative finding, i.e., failing to find an association even when a causal effect is truly present. There is no reason to automatically accept "lack of correlation" as a clear indicator of "no causal effect" any more than to accept "presence of correlation" as a clear indicator of "causal effect present." The interpretation of either result calls for a thorough assessment. An overall assessment considers the full range of studies that provide pertinent information regardless of their results and integrates the full range of relevant studies. Negative studies may reflect insufficient statistical power to detect associations due to

small populations or limited range of exposure, a particular challenge in studying rare diseases like cancer. Studies that do not measure exposure accurately are also more likely to fail to detect a true association that may be present, with the error in exposure estimation tending to shift measures of association towards the null value (showing little or no association).

Epidemiologists cannot ethically conduct experiments with controls where one group of people is intentionally exposed to a suspected toxic agent while a control group is not and then follow these groups to compare how many from each group develops a particular disease. Epidemiologists must instead study groups that have already been exposed to assess the incidence of disease in comparison to an unexposed population to determine whether those who were more highly exposed to the toxic substance have a greater risk of disease than those not exposed. Epidemiologists may study occupational exposures, where people in a particular occupation are exposed through their work to a suspected toxicant, or community exposures, which are often more difficult to study because of the challenge in measuring exposure and possible confounders that may be associated with exposure. For this reason, the C8 Health Project was unique in that it enabled the study of nearly 70,000 people whose exposure was markedly elevated in some cases and could be reconstructed given the well-defined source of contamination. The extensive data collection on this large, highly exposed population substantially advanced our understanding of the potential human health effects of elevated exposure to PFOA.

IV. PFOA EXPOSURE IN HUMANS

C8 is a name given to perfluorooctanoic acid (PFOA), a man-made chemical used in manufacturing various consumer products including non-stick cookware, protective finishes on carpets and fabrics as well as water-resistant clothing. DuPont's West Virginia Washington Works Plant in southwest Parkersburg released PFOA into the air and Ohio River from the 1950s until

the early 2000s. C8 reached drinking water supplies by entering the groundwater and was detected in six water districts near the DuPont plant in 2002. A class action lawsuit brought by the communities against DuPont resulted in a Settlement Agreement in the Wood County Circuit Court. As part of that settlement, Brookmar Inc., an independent company, was set up and conducted a year-long survey (August 2005 - July 2006) called the C8 Health Project. The C8 Health Project gathered information through interviews and questionnaires and collected blood samples from about 69,000 people living near the Washington Works plant in West Virginia. The settlement also established that a group of public health scientists would assess whether or not there is a probable link between PFOA exposure and disease in the community. The members of the Science Panel were jointly selected by the lawyers for the community and DuPont. The C8 Science Panel consisted of Dr. Tony Fletcher of the London School of Hygiene and Tropical Medicine, Dr. Kyle Steenland of Emory University in Atlanta and myself. We were chosen because of our extensive experience designing and carrying out environmental health studies and the view of the parties in the settlement that we would be able to objectively generate and evaluate the evidence. We came to the C8 Science Panel as independent epidemiologists--scientists trained in gathering information to evaluate whether environmental factors may or may not be causing disease in groups of people and remained independent and neutral throughout. The settlement paid for our work but the parties to that case did not direct what we did or how we did it. We had no preconceived notion as to whether or not C8 exposure affected human health.

The first stage of our work was compiling what was known from the research of others regarding health effects of PFOA and designing and implementing the new research needed to make an informed assessment about possible health effects. These new studies on exposure to PFOA and health were conducted in the communities in the Mid-Ohio Valley. As these studies

were completed, we shared the results with the Court overseeing the settlement, the community of the Mid-Ohio Valley, and scientists. These results became available at different times, not all at once, and were shared as they became available. The Panel emphasized the results of these studies provided background and valuable information for making an evaluation of whether there is or is not a probable link between PFOA exposure and any disease, but that evaluation was a separate phase of the Science Panel's work.

Following the research studies the next task was to make a judgment regarding the evidence of a causal link between PFOA and the risk of developing a disease. The Settlement Agreement between the plaintiffs and the defendant DuPont required the Science Panel determine whether there is or is not a probable causal link between PFOA exposure and any disease. This determination was to be based on health research carried out by the Science Panel in the Mid-Ohio Valley population exposed to PFOA, as well as other published scientific research which could help in that assessment. Once all the studies concerned with a specific disease were completed, shared with the court, and made public, we combined those findings with those of studies done by others, including laboratory research, to make our assessment of whether or not there is a probable link between C8 exposure and that illness. The research results and the assessment of whether there is a probable causal link were completed at different times for different illnesses. Our interpretation and judgment regarding the concept of “probable link” was based on the potential for a causal influence of PFOA, taking into account whether observed associations were more likely to be due to some bias or artifact versus due to a causal effect of PFOA. When we came to the conclusion that a causal effect was more likely to be responsible, even if only slightly more likely, we determined that a probable link was present.

To make this determination, we considered the full range of available evidence, including the research we conducted, epidemiologic studies previously reported, and toxicology of these chemicals. Where associations were identified, we examined the likelihood that they reflected a cause and effect relationship versus a spurious correlation due to reverse causality or some other influences on disease risk. These are standard, well-accepted principles in making a judgment about the potential for an exposure to affect risk of disease used by epidemiologists As a result of the above analyses, the C8 Science Panel came to the conclusion there Is a probable causal link between PFOA exposure and six human diseases and conditions: kidney cancer, testicular cancer, ulcerative colitis, thyroid disease, hypercholestolemia and pregnancy induced hypertension (preeclampsia). It is important to note that in performing our assigned task the C8 Science Panel was instructed to focus only on disease, not on changes in biomarkers that could potentially be used to predict future disease. For example, as a result, we analyzed whether PFOA caused the recognized condition of "hypercholesterolemia" but not whether it generally resulted in elevation of cholesterol levels that did not yet rise to the level required to diagnose hypercholesterolemia. Similarly, the C8 Science Panel did not analyze whether elevated liver enzymes levels or uric acid levels were associated with PFOA exposure. However, other researchers have addressed these associations as described in more detail below and have concluded that there is likely to be a causal link to these elevated biomarkers as well.

For this report, I updated the research done by the panel regarding probable causal links between PFOA exposure and human disease and provide my opinions on this topic below. My opinions have been reached to a reasonable degree of scientific certainty and are based on my work on the C8 Panel, my review of the scientific literature performed before, during and after the

completion of that work[3], and my education, training and experience in the field of epidemiology. The methodology I employed here to review the scientific literature and reach the opinions below is the same methodology I use daily as an epidemiologist. I describe this methodology above in the Background on Epidemiology section.

Thyroid disease – There is support in the scientific literature for a causal link between cumulative PFOA exposure and thyroid disease, specifically hyperthyroidism and hypothyroidism. Based upon my evaluation of this research, and the collective opinion of the C8 Science Panel, it is more probable than not that exposure to PFOA is capable of causing thyroid disease in human. This causal relationship is supported by research done as part of the C8 Health Project (116)[4], with some support from the analysis of National Health and Nutrition Examination Survey (NHANES) data (74). In the analysis of the Ohio/West Virginia population, there was an association between historical PFOA exposure and increased risk of both hypothyroidism and hyperthyroidism in women but not men. However, the prospective study which began with enrollment in the C8 Health Project and identified new cases of thyroid disease going forward found a clear positive association of PFOA with hypothyroidism in men and a somewhat weaker association with hyperthyroidism in men. For hypothyroidism in women, there was a clear dose-response gradient, with the first indication of an increased risk in the third quintile of exposure which became larger in the higher exposure groups. For hyperthyroidism in woman, a dose-response relationship was found with an increase in incidence being found starting in the second quintile and continuing to rise with increasing exposure. For prospective cases (diagnosed after PFOA was measured), hypothyroidism among men increased starting in the third quintile and

[3] A Bibliography of the published articles on this topic I reviewed is attached as Exhibit B.
[4] Parenthetical numerical references are to articles in listed in Exhibit B.

showed a consistently increasing risk with increasing exposure above that level, rising to a two-fold increased risk in the uppermost quintile.

A study of thyroid disease in children in the same population also found a higher risk of thyroid disease, largely hypothyroidism, associated with measured PFOA exposure (120). A recently published study from a community in Sweden with elevated exposure to PFOS and PFHxS, with modest elevation in PFOA, did not find increased risk of thyroid disease (Andersson et al., 2019). Studies of changes in thyroid hormone levels have been mixed, with some indicative of changes in various thyroid hormones and others not (121). Although the research has been somewhat variant in regard to hypothyroidism vs. hyperthyroidism, decreased or increased thyroid hormones levels, and associations for men and women, the overall body of evidence indicates an elevated risk of thyroid disorders resulting from elevated levels of PFOA.

Ulcerative Colitis - Increasing levels of PFOA are associated with increased risk of developing ulcerative colitis based on a series of studies conducted by the C8 Science Panel. Thus, it is my opinion, and the collective opinion of the C8 Science Panel, that it is more probable than not that exposure to PFOA is capable of causing ulcerative colitis. Epidemiologic studies from the C8 Science Panel, with results from the combined community and occupational cohort (99) and from the study of disease incidence in DuPont workers (100) clearly demonstrates this association. In the first study, there was a strong dose-response gradient of increasing risk with increasing cumulative exposure. Using a cumulative exposure measure of nanograms per milliliter (ng/mL), quartiles of the distribution were examined and each of the upper three quartiles was compared to the lowest. Exposures >158 ng/ml were associated with increasing risk and the risk continued to rise with more elevated exposure. Other approaches to evaluating exposure were considered, with varying details, but all tending to show increased risk above the lowest quartile

of exposure. The study of DuPont workers (100) had more limited numbers of cases (28 in total) but also supported the presence of a positive association. A recent report from Sweden that addressed a different constellation of PFAS exposure as noted for thyroid disease (markedly elevated PFOS and PFHxS, modestly elevated PFOA) did not find increased risk of ulcerative colitis (122). Nonetheless, based on the strong evidence from the studies in Ohio/West Virginia, the evidence supports an effect of elevated PFOA on the risk of ulcerative colitis.

Kidney Cancer - There is consistent evidence of a strong association and dose-response relationship between PFOA exposure and kidney cancer based on the work of the C8 Science Panel and, it is my opinion, and the collective opinion of the C8 Science Panel, that it is probable exposure to PFOA is capable of causing kidney cancer. This opinion is based on three different studies all conducted as part of the C8 Science Panel research in the Ohio/West Virginia area. The studies consist of a geographic study by Vieira et al. (112), an occupational study of mortality DuPont workers by Steenland and Woskie (98), and a cancer incidence study that combined occupational and community cohorts by Barry et al. (6). Although there is some overlap in the populations, the methods and coverage are different enough to consider these somewhat independent of one another. In the geographic study, kidney cancer was elevated only in the Little Hocking and Tuppers Plains water districts, but not in the exposed water districts more generally compared to nearby counties. The association that was restricted to the most highly exposed water districts is a form of a dose-response gradient. Using estimated serum levels (assuming a 10-year residence in the current water district there is a clear gradient, with risk increasing above around 30 ug/l. Smoking information was not available in this study. In the occupational mortality study of DuPont workers (98), kidney cancer mortality was examined, with and without lags (in which the most recent exposure is ignored to focus on past exposures). Across the quartiles of exposure,

each compared to a population consisting of Appalachian DuPont workers at other plants, the standardized mortality ratio (relative risk) generally increased with increasing exposure. Analyses assuming 10 and 20 year lags showed the same pattern – an increased risk of kidney cancer death in the highest exposure group, which was >2700 ppm-years for the unlagged exposure. Smoking data was not available for adjustment in this study. Finally, the combined community and worker study of cancer incidence (6) integrated the strongest features of the previous studies, looking at incident cases rather than deaths, accounting for individual exposure histories, and adjusting for cigarette smoking unlike the other studies. Comparing the 2nd, 3rd, and 4th quartiles to the first quartile as the referent category, the relative risk was found to increase with increasing level of exposure . The increase in risk of kidney cancer incidence began around a cumulative exposure of 812 ng/ml-yr. Only one of the studies adjusted for smoking but there is little reason to suspect strong confounding given the source of the exposure. A study of highly exposed 3M workers (123) did not find elevated risk of kidney cancer, but had limited power due to the small study size. While the evidence indicating an effect of PFOA on kidney cancer has not become stronger subsequent to the reports of the C8 Science Panel, it remains solid based on those studies.

Testicular Cancer – The epidemiological literature generated by the C8 Science Panel supports an association between PFOA exposure and an increased risk of developing testicular cancer. It is my opinion, and the collective opinion of the C8 Science Panel, that it is more probable than not that exposure to PFOA is capable of causing testicular cancer. There are two studies that address PFOA and testicular cancer, one a geographic study in Ohio and West Virginia (111) and the other the study of the combined community and occupational cohort by the C8 Science Panel (6). In the geographic study, the numbers of cases were limited, making the results imprecise. Only one of the districts, Little Hocking, showed an elevated risk. In the examination of estimated

PFOA serum levels, the relative risks for low, medium, and high exposure groups were all below 1.0 and highly imprecise, with evidence of elevated risk in the very high group. While there was not a gradient of risk across the exposure range considered, the isolation of elevated risk in the highest exposure group is of note. The community and occupational cohort study (6) included 32 reported incident cases of testicular cancer of which 19 were validated. Across the range of exposure, there was an increasing risk of testicular cancer per log unit change in cumulative PFOA and across quartiles of exposure. Similar results were found with a 10-year lag. These two studies are consistent with an elevated risk of testicular cancer associated with increased levels of PFOA exposure. Based on the Barry et al. (6) study, the elevated risk begins above 812 ng/ml-yr cumulative exposure but this estimate is imprecise because of the rarity of this form of cancer.

Uric Acid Levels - There is rather clear and convincing evidence that higher levels of PFOA are associated with higher levels of serum uric acid. Thus, it is my opinion that it is more probable than not that exposure to PFOA is capable of causing increased uric acid levels. This is seen in the analyses of the C8 Health Project participants (97), with notable increases in average serum uric acid levels and the risk of being above the cut point defining hyperuricemia (significantly elevated serum uric acid) across the spectrum of PFOA exposure. The increase in risk was especially strong in the lower range and reflects somewhat of a ceiling effect with less of an increase across the highest levels. An elevation in risk was clear in going from the first to the second quintile of exposure, above 11.4 ng/ml of PFOA and increasing modestly with higher exposures. Evidence of this association was corroborated in studies in children (38; 84) and adults (13; 42) in other populations. While studies based on serum markers of PFOA and serum uric acid are susceptible to spurious associations due to kidney function, this is not plausible in studies based on variation in water levels as done by the C8 Science Panel.

Hyperlipidemia (high cholesterol) – A substantial number of studies have found clear associations between PFOA exposure and both total and LDL cholesterol (124). It is my opinion based on these studies that it is more probable than not that exposure to PFOA causes an increase in both total and LDL cholesterol. A preponderance of studies shows a positive association between PFOA and elevated levels of total cholesterol and LDL cholesterol, but this is not universal across studies, some of which show no association with either total or LDL cholesterol or both. Again, generalizing across a large body of studies, the most consistent and compelling association would be with total cholesterol in part because more studies have addressed this measure. This association is found in adults, children and adolescents, and pregnant women with some consistency. While an increase in average lipid levels with increasing PFOA means it is likely that hypercholesterolemia, generally defined as a total cholesterol >240 mg/dL or LDL cholesterol >110 mg/DL, will also be increased, there are fewer studies of hypercholesterolemia because much larger study populations are required. Using cross-sectional data from the C8 Health Project, Steenland et al. (95) found clear evidence that higher levels of PFOA are associated with greater risk of hypercholesterolemia, with increasing odds ratios across exposure quartiles and with a similar pattern for LDL cholesterol. In an analysis of the community and worker cohort developed by the C8 Science Panel, Winquist and Steenland (115) again found increased risk of hypercholesterolemia when compared to the lowest quintile. An association with hypercholesterolemia was also found in National Health and Nutrition Examination Survey (NHANES) data (38) where an increased risk of elevated levels of LDL cholesterol was also found. There is a strong empirical basis for concluding that higher levels of PFOA are associated with higher levels of total and LDL cholesterol, and that PFOA is associated with increased risk of hypercholesterolemia. Further support for a causal effect comes from a demonstration that the

magnitude of decline in PFOA levels was associated with the magnitude of decline in serum cholesterol and LDL in the C8 Science Panel study population (125).

An important point to note, which may explain some of the inconsistency in the findings across studies, is that the dose-response gradient shows a rapid increase in total cholesterol in the lower range of PFOA exposure but appears to plateau, with little increased risk as exposure rises further. This was true in the cross-sectional study (96) and even clearer in the cohort study in which risk increased from the first to second quintiles of PFOA exposure but did not increase further across the highest four quintiles (115). Highly exposed populations such as occupational cohorts do not consistently report associations of PFOA with cholesterol, possibly because all those studied are in the relatively high exposure range, whereas community studies of background exposure ranges more consistently identify an association. Focusing specifically on HDL cholesterol, which is inversely related to cardiovascular disease risk (higher HDL cholesterol predicts a lower risk of cardiovascular disease), fewer studies have examined the association with PFOA. The expected effect of PFOA would be to reduce HDL cholesterol levels and this has been found in some studies. In the occupational health literature, a negative association between PFOA and HDL cholesterol was found by Olsen and Zobel (67) and Wang et al. (112), but not in a number of comparable studies (e.g., 81; 86; 13). The community studies are likewise mixed in regard to an association with HDL, with the cross-sectional study of C8 Health Project participants not showing an association between PFOA and HDL cholesterol in adults (96) or children (34). An absence of association was reported for studies in community populations with background exposure (77; 31; 30; 39). One study of children did find a reduction in HDL cholesterol with higher PFOA levels (118), and another study found higher HDL cholesterol levels with increasing exposure to PFOA during pregnancy (95). Thus, the inverse association between PFOA exposure

and lowered HDL cholesterol is less clear than the positive association with total cholesterol and LDL cholesterol.

Elevated Liver Enzymes – There is support in the scientific literature for an association between PFOA exposure and elevation of at least some liver enzymes in the blood serum, reviewed recently by Knutsen et al. (2018) (124). It is my opinion that it is more probable than not that exposure to PFOA is capable of causing an increase in liver enzyme levels in the blood. A substantial number of studies have examined the correlation between serum levels of PFOA and an array of liver enzymes. Those that are most frequently studied include ALT (alanine transferase), ALP (alkaline phosphatase), AST (aspartate aminotransferase), GGT (gamma gluatmyl transferase), bilirubin (total and direct), and CCK (cholecystokinin). Many of the studies examine the entire panel of routinely assayed liver enzymes and others do so selectively. Given the large number of enzymes and large number of studies, there are an array of results which are not entirely consistent but with some patterns present. Elevated liver enzymes usually do not indicate the presence of chronic liver disease but more often some reversible cause such as inflammation or injury to the liver that has caused leakage of liver enzymes into the bloodstream. Often elevation in liver enzymes is caused by medications (over the counter or prescription), drinking alcohol, or underlying disease such as hepatitis or heart failure. The most consistent finding is an association of PFOA with increased levels of ALT, observed in the C8 Science Panel research (36; 17) in the National Health and Nutrition Examination Survey (68; 42), and in some of the occupational studies (82; 86; 13). While some other studies found no association, there is a clear weight of evidence in support of a positive association of PFOA with elevated ALT. Perhaps the next most commonly observed association is with PFOA and elevated GGT, found in some occupational studies (82; 86) and in the National Health and Nutrition Examination Survey (68;

42) but not in the C8 Science Panel research. Beyond that, the findings for all the other enzymes, including AST and bilirubin as the most frequently studied, are not supportive of an association with PFOA.

Immune System Effects - Several studies support an association between PFOA exposure and immune response, particularly from prenatal exposures and subsequent immune response in children (Rappazzo et al., 2017; Liew et al., 2018) (128; 129). Based upon these studies, it is my opinion that it is probable that exposure to PFOA can affect the immune response to pathogens. While there are a number of studies of indicators of PFOA and immune function, fewer studies considered PFOA exposure and actual infectious disease. In a study of prenatal exposure and early childhood illness in Denmark (29), no association was observed overall or for boys alone, but for girls, there was a gradient of increasing risk of infectious disease with a clear dose-response gradient across quartiles of PFOA. Another Danish study (14) evaluated prenatal PFOA levels in relation to infectious diseases among children ages 1-4. Across three levels of PFOA exposure, there was a gradient of increasing risk for fever, but not for cough, nasal discharge, diarrhea, or vomiting. A study from Japan (80) had small numbers that limited ability to examine the one infectious disease considered, otitis media (ear infection) for which they found an adjusted odds ratio showing increased incidence. Self-reported influenza infections and colds among participants in the C8 Health Project did not indicate an association between PFOA level and risk of these diseases (71). In the National Health and Nutrition Examination Survey data, a positive association was found for PFOA and risk of rhinitis but a negative association for the risk of mumps and rubella (105). Although the numbers of cases were small, a Norwegian study reported a modest association between PFOA and episodes of colds and gastroenteritis and a negative association with rubella antibodies (36). More recent studies continue to yield mixed findings

(Impinen et al., 2018, 2019) (130;129). The possibility of PFOA being associated with increased risk of infection is supported indirectly by some research suggesting elevated levels of PFOA are associated with a weaker response to influenza vaccination (57), though another study noted a more favorable response to influenza vaccination with higher PFOA levels (106). Several studies have reported a decreased response to vaccine to prevent diphtheria (a bacterial respiratory disease) associated with higher PFOA levels (43; 61; 45). It is difficult to draw any firm conclusions given the diversity of conditions examined and inconsistent results. It seems plausible, perhaps even likely, that there is some increase in infections in relation to PFOA serum levels, but the research does not provide clear or consistent evidence of increased risk of specific infectious diseases associated with elevated PFOA. .

Preeclampsia, Pregnancy Induced Hypertension – There is some evidence in the published literature for an association between PFOA exposure and the incidence of preeclampsia or pregnancy induced hypertension. Our study of the C8 community showed an increased risk for preeclampsia. (89). Another study of this population showed a weak association between PFOA exposure and pregnancy induced hypertension (16). Attempts at replicating these findings have not been supportive (Huang et al., 2019; Wikstrom et al., 2019) (126; 127) (calling this association into question given the modest increases found in the C8 Science Panel studies and non-supportive evidence in subsequent studies.

In addition to the above adverse health effects that I believe more probably than not are related to PFOA exposure, there are a number of other health conditions that are under study and may reach this threshold in the future. The health effects discussed below fall into this category.

Prostate Cancer – There is limited evidence supporting an association between PFOA exposure and risk of prostate and ovarian cancers. In the study by Hardell et al. (51), the

association between PFOA and prostate cancer was divided by family history. Since family history predisposes to prostate cancer at a younger age, this has indirect relevance to an age-specific effect. They did find that PFOA above the median was associated with increased risk of prostate cancer in the subgroup with a family history of prostate cancer. In a study of 3M workers, Raleigh et al. (2014) (123) did not find an association between PFOA and prostate cancer whereas in a study of DuPont workers, Steenland et al. (2015) (132) reported a trend towards higher risk of medically confirmed prostate cancer with higher levels of PFOA exposure. The relationship of PFOA to prostate specific antigen (PSA) was examined by Ducatman et al. (22) and stratified by age, with weak evidence that there was a positive association among younger men (20-49). PSA level is considered a marker for the development of prostate cancer, although the accuracy of this marker has been questioned in recent years.

Ovarian Cancer – There is limited evidence supporting an association between PFOA exposure and ovarian cancer. The only evidence addressing PFOA and ovarian cancer comes from the geographic study by Vieira et al. (111) and the analysis of combined community and occupational cohort by Barry et al. (6). Vieira et al. (111) identified 48 cases of ovarian cancer and found elevated risks in the Little Hocking and Belpre water districts. Examining estimated serum levels of PFOA, and dividing the population into quartiles, there was evidence of an association. In contrast, the cohort study (6) which included 43 confirmed cases found no association with a continuous measure of PFOA exposure.

It is important to note that as more research is conducted on PFOA exposed populations, more evidence has accumulated suggesting associations between PFOA and human illness.

Because drinking water has only recently become a focus of attention for PFOA contamination and because a testing of both public and private drinking water sources had detected significant levels of PFOA in many locations across the United States, it is highly likely that more research will be done that may add to support for an association between PFOA and adverse human health effects in the future.

Based upon my research, specifically including my work on the C8 Science Panel, my review of the medical, scientific and epidemiological literature, as well as my education, training and experience as an epidemiologist, it is my opinion to a reasonable degree of scientific certainty that elevated PFOA exposure increases the risk of the development of certain diseases and conditions referenced above. The question of a lower limit for this effect is not resolved at present, but there is evidence that for exposures above background levels, elevated risks are likely to be present, particularly for developmental immune disorders but possibly for other conditions[5] Even at current US "background" levels, studies have repeatedly suggested biological effects on the immune system with negative effects being seen with increasing PFOA blood levels. Studies of Norwegian children (46), a study from the Danish National Birth Cohort (29) and a study of children in the Faroe Islands (43) have all shown negative immune response with increasing PFOA blood levels at or near U.S. background levels. Because PFOA demonstrates adverse biological effects even near "background" levels, evidence does not exist for establishing a level of PFOA exposure below which no negative effects can be assured. While it is true that evidence of increased incidence of disease for some conditions listed above were only seen in the highest

[5] In the Michigan report "Scientific Evidence and Recommendations for managing PFAS Contamination in Michigan" we concluded that the current EPA health advisory limit of 70 ppt for drinking water might not be sufficiently protective because increases in ulcerative colitis, some cancers and other health effects have been reported for exposures predicted in people consuming water containing this level of PFOA. https://www.michigan.gov/documents/pfasresponse/Science_Advisory_Board_Report_641294_7.pdf, at p. 11.

exposed groups, for other outcomes such as elevated cholesterol and ulcerative colitis, increased risks were present in the near-background exposure range. It is unclear whether exposures at or below background are associated with all of the diseases causally linked to PFOA exposure, but since a dose- response relationship has emerged for a number of the associated illnesses, what is clear is that as exposure increases above background so does risk of disease.

Of note about the exposures involved in the Ohio River Valley studied in the C8 Health Project is that they varied by community to a significant extent. Little Hocking, OH had very high levels of PFOA in its municipal water supply and the population there had correspondingly higher PFOA levels in their blood. The other communities studied, Lubeck, WV, Tupper Plains, OH and Mason County, WV had variably elevated levels, much lower but still substantially elevated above background levels of PFOA in their municipal water supplies and again residents of those communities had levels of PFOA in their blood that corresponded to the levels in their drinking water source when tested (Shin et al., 2011) (133). Accordingly, the almost 70,000 people who participated in the C8 project had a wide range of exposures to PFOA and a wide range of PFOA blood levels.

In this case, the data I have seen from the NYSDOH indicates that there were two rounds of testing and some individuals were tested twice. [redacted] REDACTED PURSUANT TO PROTECTIVE ORDER (ECF NOs. 131 & 132) [redacted] This mean exposure level is lower than Little Hocking, OH and Lubeck, WV, but higher or analogous to

Belpre and Tuppers Plain, OH, and Mason County, WV that were part of the C8 Health Project (Frisbee et al., 2009) (134).[6]

I have reviewed the Cancer Incidence Investigation 1995-2014 conducted by the NYS Department of Health for the Village of Hoosick Falls in May of 2017. This report provides data on cancer incidence in the Hoosick Falls community. Such information is routinely collected by the state cancer registry and can be used for general surveillance purposes. It is not designed to be nor is it useful for etiologic studies of the potential effect of an environmental toxicant on diseases in the population. There are several reasons that it is not suitable for such purposes: 1) There is no exposure information other than that the person resided in a community with elevated levels of PFOA in the water at the time of diagnosis with no information on how long they resided in that community, and no direct information on the levels of PFOA in the water over the period that the person lived there or even a basis for estimating cumulative PFOA exposure. For example, if someone were exposed to the elevated levels of PFOA in the water and moved prior to diagnosis, such cases would not be included in the tabulation; 2) There is no information on other potential causes of these cancers that may need to be taken into account to isolate any effect of PFOA, which might mask true associations or generate spurious associations; 3) The numbers of events for the cancers of particular interest are simply too small to be informative. As a scientific contribution to the previously conducted studies examining potential health effects of PFOA exposure, there is no added value to this analysis. It is entirely reasonable to tabulate and share the data as a general

[6] Participation in the C8 Health Project required that minimum 12 month exposure to drinking water of 500 ppt or greater. NYSDOH had no such requirement and people in the Town of Hoosick who did not drink from a contaminated well were likely included in those tested. The class definition in this case requires that the person with PFOA in their blood above 1.86 ug/L also consumed contaminated drinking water from a private well or the Hoosick Falls municipal water system. Therefore, comparison of the mean blood levels in the C8 Health Project communities to the subset of people in the Town of Hoosick who meet the class definition is more appropriate than comparing the mean of all people tested to the C8 Health Project community levels. Moreover, because we do not know at this point how many of the people above 1.86 ug/L in the NYSDOH testing would not meet the class definition, the mean likely understates the true class mean once the class is identified.

description of the community's health experience but it simply is not suitable for addressing cause and effect relationships in this population or more generally.

My approach with the C8 Panel and in coming to the opinions stated herein was based upon generally accepted principles practiced in the field of epidemiology and that my opinions regarding the causal link between PFOA exposure and human health effects are generally accepted in my field. My opinions and conclusions are also supported by the June 2018 Draft Toxicological Profile for Perfluoroalkyls which states: "The available epidemiology studies suggest links between perfluoroalkly exposure and several health outcomes.." listing hepatic effects, cardiovascular effects, endocrine effects, immune effects, reproductive effects and developmental effects linking PFOA exposure in each of these adverse health outcomes.[7] The 2018 report by the Health Effects Subcommittee of the New Jersey Water Quality Institute supporting the lowering of the maximum amount of PFOA that should be permitted in drinking water to 14 ppt. also succinctly states what I believe be the consensus view of epidemiologists and public health experts about PFOA:

> In summary, associations of PFOA with numerous health endpoints have been found in human populations with evidence supporting criteria for causality for some endpoints. These health endpoints include both non-carcinogenic effects in the general population and both non-carcinogenic effects and cancer in communities with drinking water exposure. The epidemiologic data for PFOA are notable because of the consistency between results among human epidemiologic studies in different populations, the concordance with toxicological findings from experimental animals, the use of serum concentrations as a measure of internal exposure, the potential clinical importance of endpoints for which associations are observed, and the

[7] https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf, at p. 25.

observation of associations within the exposure range of the general population.[8]

12/28/19
Date

DAVID A. SAVITZ, Ph.D.

[8] https://www.state.nj.us/dep/watersupply/pdf/pfoa-appendixa.pdf, Executive Summary, pp. 8-9; See also, https://www.michigan.gov/documents/pfasresponse/Science_Advisory_Board_Report_641294_7.pdf.

EXHIBIT A

CURRICULUM VITAE

September 2019

David A. Savitz, Ph.D.
Providence, RI 02912
401-863- 6090(Phone) 401-863-3713(Fax)
David_Savitz@Brown.edu

EDUCATION

BA, **Brandeis University,** Waltham, Massachusetts - 1971-1975

Ohio State University College of Medicine, Columbus, Ohio
(Passed Part I, National Boards) - 1975-1976

MS, **Ohio State University,** Department of Preventive Medicine
Columbus, Ohio - 1976-1978

PhD, University of Pittsburgh, Department of Epidemiology
Graduate School of Public Health
Pittsburgh, Pennsylvania - 1979-1982

HONORS/AWARDS

1975 - Highest Honors in Psychology
Brandeis University
B.A., summa cum laude
Brandeis University

1983 - Excellence in Teaching Award, Second Year Medical Students, University of Colorado School of Medicine

1999 - Elected to American Epidemiological Society

William R. Gemma Award, Outstanding Alumnus, Department of Preventive Award, Ohio State University

2003 - Slone Memorial Lecturer, Slone Epidemiology Center at Boston University

2004 - Advancing Knowledge Award, Coalition for Excellence in Maternal and Child Health Epidemiology

Distinguished Graduate Award, University of Pittsburgh Graduate School of Public Health

2007 - Elected Member, National Academy of Medicine (formerly Institute of Medicine)

2011 - Distinguished Lecturer, Occupational and Environmental Epidemiology Branch, National Cancer Institute

ACADEMIC APPOINTMENTS

2010–Present	Professor of Epidemiology School of Public Health Brown University Providence, Rhode Island
2010–Present	Professor of Obstetrics and Gynecology The Warren Alpert Medical School Brown University Women and Infants Hospital Providence, Rhode Island
2018-Present	Professor of Pediatrics The Warren Alpert Medical School Brown University Women and Infants Hospital Providence, Rhode Island
2018-Present	Associate Dean for Research School of Public Health Brown University Providence, Rhode Island
2013-2017	Vice President for Research Brown University Providence, Rhode Island
2006-2010	Charles W. Bluhdorn Professor of Preventive Medicine Director, Disease Prevention and Public Health Institute Mount Sinai School of Medicine New York, New York
2003-2005	Cary C. Boshamer Distinguished Professor Department of Epidemiology School of Public Health University of North Carolina Chapel Hill, North Carolina
1999-2005	Associate Director, Center for Infectious Diseases
1996-2005	Professor and Chair
1993-1996	Professor
1992-2005	Member, Lineberger Comprehensive Cancer Center
1989-1992	Associate Professor
1986-2005	Fellow, Carolina Population Center
1985-1989	Assistant Professor
1981-1985	Assistant Professor Department of Preventive Medicine and Biometrics University of Colorado School of Medicine Denver, Colorado
1979-1981	Public Health Service Trainee in Psychiatric Epidemiology

Department of Epidemiology
Graduate School of Public Health
University of Pittsburgh
Pittsburgh, Pennsylvania

1977-1979 Researcher (Epidemiology)
Ecology and Ecosystems Analysis Section
Battelle-Columbus Laboratories
Columbus, Ohio

OTHER PROFESSIONAL APPOINTMENTS

Elected Positions in Professional Societies

Society for Epidemiologic Research

1987-1991	Secretary-Treasurer
1994-1997	Executive Committee Member
1999-2000	President-Elect
2000-2001	President
2001-2002	Past President

International Epidemiological Association

1996-2001	North American Regional Councilor

Society for Pediatric and Perinatal Epidemiologic Research

2003-2004	President-Elect
2004-2005	President

International Society for Environmental Epidemiology

2012-2014	Executive Council Member

Appointed Membership to Editorial Boards

1988-1990, 2010-Present	Associate Editor, American Journal of Epidemiology
1990-1998	Editor, American Journal of Epidemiology
1989-1990	Editorial Board, Bioelectromagnetics
1993-1997, 2009-Present	Editorial Board, Environmental Health Perspectives
1996-2001	Editorial Board, Japanese Journal of Epidemiology
1998-2005	Editorial Board, Pediatric and Perinatal Epidemiology
2001-2013	Editor, Epidemiology
2005	Editorial Board, Annual Reviews in Public Health
2008-2012	Editorial Board, Journal of Neurodevelopmental Disorders

Manuscript Review	American Industrial Hygiene Association Journal
	American Journal of Epidemiology
	American Journal of Industrial Medicine
	American Journal of Obstetrics and Gynecology
	American Journal of Preventive Medicine
	American Journal of Public Health
	Annals of Epidemiology
	Bioelectromagnetics
	Birth Defects Research A

British Journal of Obstetrics and Gynecology
CA -- A Cancer Journal for Clinicians
Cancer Epidemiology Biomarkers and Prevention
Cancer Causes and Control
Cancer Research
Critical Reviews in Toxicology
Developmental Origins of Health and Disease
Drug Safety
Environment International
Environmental Health Perspectives
Environmental Research
Environmental Technology Letters
Epidemiology
Ethnicity and Diseases
International Journal of Epidemiology
Journal of the American Medical Association
Journal of Clinical Epidemiology
Journal of Exposure Analysis and Environmental Epidemiology
Journal of the National Cancer Institute
Journal of Occupational and Environmental Medicine
Journal of Pediatrics
Journal of Toxicology and Environmental Health
Journal of Urban Health
Mayo Clinic Proceedings
New England Journal of Medicine
Obstetrics and Gynecology
Occupational and Environmental Medicine
Occupational Hygiene
Pediatric and Perinatal Epidemiology
Pediatrics
PLOS One
Preventive Medicine
Reproductive Toxicology
Risk Analysis
Sleep
Teratology

Intramural/Extramural Committees

University of Colorado

1982-1983	Member, Faculty Senate
1982-1985	Member, Admissions Committee Department of Preventive Medicine and Biometrics
1983-1985	Chairperson, Comprehensive Examination Committee Department of Preventive Medicine and Biometrics
1983-1985	Member, Appointments and Promotions Committee Department of Preventive Medicine and Biometrics
1984-1985	Director, Epidemiology Program Community Health Section Department of Preventive Medicine and Biometrics

University of North Carolina

1986-1992	Chair, Seminar Committee

	Department of Epidemiology
1986-1989	Member, Institutional Review Board on Research Involving Human Subjects School of Public Health
1986-1991, 1994-1996	Member, Admissions Committee Department of Epidemiology
1987-1996	Member, Training Committee Carolina Population Center
1987-1990	Co-Chairperson, Low Birth Weight Prevention Task Force, Center for Health Promotion and Disease Prevention
1988-1990	Member, Seminar Committee Carolina Population Center
1989-1990	Alternate Member, University Faculty Council
1989-1994	Advisory Council, Carolina Population Center
1989	Member, Search Committee, Nutrition Department Chair
1990	Member, Strategic Planning Task Force School of Public Health
1991-1993	Member, Academic Programs Committee School of Public Health
1993-1996	Member, Appointments and Promotions Committee School of Public Health
1994-1996	Member, Advisory Committee for the Center for Environmental Medicine and Lung Biology
1996- 2004	Member Advisory Board, Sheps Center for Health Services Research
1996-1997	Member, Search Committee, Director of Lineberger Cancer Center
1997-1998	Chair, Search Committee, Department of Biostatistics Chair
2002-2003	Chair, Search Committee, Department of Maternal and Child Health Chair
2004-2005	Chair, Advisory Board, Sheps Center for Health Services Research
2005	Chair, Search Committee, Department of Nutrition Chair

Brown University

2010-2011	Member, Admissions Committee
2010-2012	Chair, Search Committee, Environmental Epidemiology Faculty Recruitment
2012	Chair, Methods Curriculum Review Committee
2018-Present	Associate Director, Center for the Study of Children at Risk
2018-Present	Chair, Internal Advisory Committee, Advance-CTR

Grant Review

1983	Grant Review, American Cancer Society New York, New York
1983-1989	Grant Review, National Science Foundation Washington, D.C.
1984, 1986	Grant Review, March of Dimes
1985, 1988, 1991, 1993	Member, Special NIH Study Section

1986-1987	Grant Review, Electric Power Research Institute
1989	Grant Review, National Institute of Environmental Health Sciences
1989	Grant Review, Health Effects Institute
1989-1992	Member, March of Dimes Research Advisory Committee on Reproductive Hazards in the Workplace, Home, Community and Environment
1990, 1992, 1994	Grant Review, Health and Welfare Canada
1990	Grant Review, U.S. Department of Energy
1991-1993	Special Reviewer, Epidemiology and Disease Control I Study Section, National Institutes of Health
1994-1998	Charter Member, Epidemiology and Disease Control I Study Section, National Institutes of Health
1994	Grant Review, Dutch Cancer Society
1998	Special Reviewer, Radiation Epidemiology Branch National Cancer Institute
1999	Special Reviewer, Occupational Epidemiology Branch National Cancer Institute, Johnson Foundation
1989-1992	Member, March of Dimes Research Advisory Committee on Reproductive Hazards in the Workplace, Home, Community and Environment
1994-1998	Charter Member, Epidemiology and Disease Control I Study Section, National Institutes of Health
2005	March of Dimes Birth Defects Foundation, Social and Behavioral Sciences Review Committee
2005	Young Epidemiology Scholars Program, Robert Ward
2012	Reviewer, Autism Networks and Center Programs Study Section, National Institutes of Health
2012	Ad Hoc Member, Infectious, Reproductive, Asthma, and Pulmonary Conditions Study Section, National Institutes of Health
2012	Reviewer, Core Infrastructure and Methodological Research for Cancer Epidemiology Cohorts Study Section
2012	Chair, Contract Review, Exposure to Contaminants in the Generation R Study, National Institute of Environmental Health Sciences
2014-2019	Member, National Institutes of Health Grant Review, Conflict/Special Study Sections (multiple)
2016, 2017	Chair, GULF Synthesis Grants Review Committee, GULF Research Program, National Academies of Sciences, Engineering, and Medicine

Other Scientific Review Committees, Congressional Testimony, and Related Professional Activities

1982-1983	Member, Planning Committee Cancer Prevention Conference AMC Cancer Center and Penrose Hospital
1983	Member, Governor's Advisory Group on Rocky Flats, Colorado State Government
1984	Member, Extremely Low Frequency Electromagnetic Fields Bioeffects Review Committee American Institute for Biological Sciences
1985-1986	Expert Witness, Benzene Regulation Hearings

	U.S. Occupational Safety and Health Administration
1986-1988	Member, Family Health International Protection of Human Subjects Committee
1986	Member, Electric Power Research Institute Program Review on Non-Ionizing Radiation
1986-1991	Member, National Council on Radiation Protection and Measurements Scientific Committee 79 Extremely Low Frequency Electric and Magnetic Fields
1987	Testimony, U.S. House of Representatives Subcommittee on Water and Power Resources and Human Development Planning Conference
1988	Participant, National Institute for Child Health on the Reproductive Effects of Video Display Terminal Use
1988	Member, Ad Hoc Committee on Reproductive and Developmental Epidemiology U.S. Environmental Protection Agency
1989-1992	Member, Peer Review Committee Woburn Environment and Birth Study
1989-1992	Chair, Working Group on Electromagnetic Fields Environmental Epidemiology Planning Project Health Effects Institute
1990	Member, Planning Committee for International Symposium on Birth Defects Epidemiology March of Dimes Birth Defects Foundation
1993-1996	Vice Chairman, Committee on Possible Effects of Electromagnetic Fields on Biologic Systems National Research Council, National Academy of Sciences
1994-1996	Member, Committee to Review the Health Consequences of Service During the Persian Gulf War, Institute of Medicine, National Academy of Sciences
1997-2001	Member, Maternal and Fetal Medicine Network Advisory Committee, National Institute of Child Health and Human Development
1999	Member, Panel of Court Appointed Scientific Experts (CASE) for the American Association for the Advancement of Science (AAAS)
1999-2004	Member, Board of Scientific Counselors, National Cancer Institute
1999	Co-Chair, Scientific Organizing Committee, NIEHS Conference on Epidemiology in the Twenty-First Century
1999-2000	Member, External Advisory Board, The University of Iowa College of Public Health
2000-2002	Member, American Cancer Society Breast Cancer Prevention Forum
2000-2013	Member, Standing Committee on Epidemiology, International Commission on Non-Ionizing Radiation Protection
2001	Witness, Senate Cancer Coalition, Hearing on Cancer Clusters
2001-2005	Member, Advisory Committee for the Trucking Industry Particle Study, Harvard University

2002	Panel Member, Joint Science, Technology, and Law Program and American Law Institute, National Academy of Sciences
2002	Panelist, Health Canada Workshop Held to Identify Critical End Points for Assessment of the Health Risks Related to Trihalomethanes in Drinking Water
2002-2003	Member, Expert Panel on Risks and Benefits of Policies to Reduce Human Methyl Mercury Exposure through Fish Consumption, Harvard Center for Risk Analysis
2002-2003	Member, Advisory Panel on Health Effects of Blood Lead Levels <10μg/dl in Children, Centers for Disease Control and Prevention, National Center for Environmental Health
	Consultant, Case-Control Study of Gynecologic Cancers in Northern Vietnam, Family Health International
2002 – 2012	Member, Agricultural Health Study Advisory Committee
2004 - 2005	Armed Forces Epidemiologic Board
2004	Chair, National Children's Study Sampling Design Workshop
2004-2005	Member, Committee on EPA's Exposure and Human
2004-2006	Health Reassessment of TCDD and Related Compounds, Board on Environmental Studies and Toxicology, National Academy of Sciences
2005-2006	Member, Committee on Preterm Birth: Causes, Consequences, and Prevention, Institute of Medicine, National Academy of Sciences
2006—2009	Member, Advisory Panel on Research, Association of American Medical Colleges
2006 – 2010	Member, University of North Carolina School of Public Health External Advisory Board
2006– 2008	Chair, Committee on Making the Best Use of the Agent Orange Exposure Reconstruction Model, Institute of Medicine, National Academy of Sciences
2007 – 2009	Chair, Committee on Contaminated Drinking Water at Camp Lejeune, National Research Council, National Academy of Sciences
2008 – 2009	Member, Committee on Reexamination of IOM Pregnancy Weight Guidelines, Institute of Medicine, National Academy of Sciences
2009	Member, International Agency for International Research on Cancer, Monograph 100 Working Group
2010 – 2011	Member, Committee on Obesity Prevention Policies for Young Children, Institute of Medicine, National Academy of Sciences
2010	Member, Planning Committee for Institute of Medicine Workshop on Assessing the Effects of the Gulf of Mexico Oil Spill on Human Health
2012 – 2014	Member, Centre for Research in Environmental Epidemiology Scientific Advisory Committee
2015 – Present	Co-Chair, ISGlobal (formerly Centre for Research in Environmental Epidemiology [CREAL]) Scientific Advisory Committee

2013-2014	Chair, Committee to Review of EPA's Draft Paper on State of the Science on Nonmonotonic Dose Response, National Research Council
2013	Member, Advisory Council, Population Sciences and Epidemiology Integrated Review Group Evaluation, Center for Scientific Review, National Institutes of Health
2014-2016	Member, Board on the Health of Selected Populations, Institute of Medicine, National Academy of Sciences
2015	Chair, World Health Organization Workshop on the Effect of Maternal Influenza and Influenza Vaccination on the Developing Foetus, Montreal, Canada
2015-2017	Chair, Committee to Assess the Department of Veterans Affairs Airborne Hazards and Open Burn Pit Registry, National Academy of Medicine
2017-2018	Member, Committee on the Review of the Health Effects of Electronic Nicotine Delivery Systems (ENDS), National Academy of Medicine
2017-Present	Academic Advisor, Michigan PFAS Action Response Team
2018-Present	Chair, Research Committee, Health Effects Institute
2019-Present	Chair, Committee to Review the Long-Term Effects of Antimalarial Drugs, National Academy of Medicine

TRAINING RECORD AS PRIMARY ADVISOR TO MASTERS AND DOCTORAL STUDENTS

Masters

University of North Carolina

1988	Lisa Feingold, MSPH
	Peter S. Kapernick, MPH
1989	Sally S. Harris, MPH
1990	Michael T. O'Shea, MPH
	Sara M. Sarasua, MSPH
1991	Kathryn M. Menard, MPH
1992	Josephine A. Evans, MPH
1996	Kurtis Andrews, MSPH
	Michael Gallagher, MSPH
	Gayle Shimokura, MSPH
1997	Valerie King, MPH
2004	Nora Franceschini, MPH
	Yevgeniy Sheyn, MPH
2005	Cherrie Heller, MPH
2010	Michele La Merrill, MPH

Brown University

2012	Paul Davis, MPH
	Hannah Shamjii, MPH

Doctoral

University of North Carolina

1987	Hillary Klonoff-Cohen, PhD
1989	Debora Barnes, PhD
1990	Ester John, PhD
1991	Katherine M. Brett, PhD
	Martha Ann Keels, PhD
	Howard Morrison, PhD
	Shao Lin, PhD
1992	Cheryl Blackmore, PhD
	Debra E. Irwin, PhD
1993	Elizabeth M. Barnett, PhD
1994	Tye E. Arbuckle, PhD
	Laurie Elam Evans, PhD
	Kristine-Anne ToloPhD
	Suzanne L. West, PhD
	Jun Zhang, PhD
1996	Cande Ananth, PhD
	Kathryn Curtis, PhD
	Linda, Kaste, PhD
1997	Linda Pastore, PhD
1998	Amy Sayle, PhD
1999	Katherine E. Hartmann, PhD
2001	Nancy Dole, PhD
	Rukmini Bagchee Balu, PhD
2002	Juan Yang, PhD
	Lisa Pompeii, PhD
2004	Sherry Farr, PhD
2005	Emily Harville, PhD

Brown University

2014	Valery Danilack
2017	Kimberly Glazer

TEACHING ACTIVITIES

University Courses

University of Colorado

1981-1985	Discussion group leader, Epidemiology course for Medical Students – 12 contact hours each year
1983	Excellence in Teaching Award, Second Year Medical Students, University of Colorado School of Medicine
1982-1984	Introduction to Occupational and Environmental Health – 30 contact hours

1984-1985	Advanced Epidemiologic Methods – 30 contact hours

University of North Carolina

1985-1996, 1998-2005	Epidemiologic Research Methods – 35 contact hours
1986-1994	Reproductive Epidemiology – 26 contact hours
1997	Advanced Epidemiologic Methods – 30 contact hours

Brown University

2011-2012	Environmental and Occupational Epidemiology – 40 contact hours
2011	Critical Epidemiology – 40 contact hours
2013, 2016-2017, 2019	Interpretation and Application of Epidemiology – 40 contact hours
2018-2019	Reproductive Epidemiology – 40 contact hours

Other Teaching

1988	New England Epidemiology Institute Summer Program Course on Occupational and Environmental Epidemiology
1991	University of Michigan Summer Program in Epidemiology Course on Environmental Epidemiology
1997	Faculty for the Society for Epidemiologic Research Student Workshop

GRANT AND CONTRACT SUPPORT

Completed

Reproductive Hazard Surveillance Among Oil, Chemical, and Atomic Workers Union Members (M. Orleans, Principal Investigator, D. Savitz, Co-Investigator) March of Dimes, $32,000, January 1, 1981-August 31, 1984.

Case-Control Study of Invasive Cervical Cancer (R.F. Hamman, Principal Investigator, D. Savitz, Project Director), National Cancer Institute through Westat Subcontract, $140,961, June 1, 1982-July 31, 1984.

Oil Shale Technology Health and Environmental Effects Risk Analysis (W. Marine, Principal Investigator, D. Savitz, Co-Investigator), U.S. Department of Energy, $20,723, September 1, 1982-August 31, 1983; $25,000, December 1, 1983-September 30, 1984.

Ethnicity and Cancer Risk in Colorado Hispanics (D. Savitz, Principal Investigator), Biomedical Research Assistance Committee, University of Colorado, $5,670, September 1, 1982-February 28, 1983.

Cancer Risk Among Oil, Chemical, and Atomic Workers Exposed to Halogenated Hydrocarbons (D. Savitz, Principal Investigator), American Cancer Society Institutional Research Grant, $5,000, October 1, 1982-December 31, 1983.

Childhood Cancer and Electromagnetic Field Exposure (D. Savitz, Principal Investigator), Health Research, Incorporated, New York State Department of Health, $391,000, December 1, 1983-August 31, 1987.

Association of Parental Occupation with Late Fetal Mortality and Low Birth Weight (D. Savitz, Principal Investigator), March of Dimes, $50,000, January 1, 1986-November 30, 1987.

Low Birth Weight among Offspring of Smokeless Tobacco Users: A Feasibility Study (D. Savitz, Principal Investigator), University of North Carolina Research Program, $1,500, January 1, 1987 - December 31, 1987.

Epidemiologic Study of Utility Workers Exposed to Electric and Magnetic Fields (D. Savitz, Principal Investigator), Electric Power Research Institute, $5,381,303, July 1, 1987 - December 31, 1997.

Adverse Pregnancy Outcomes among Cosmetologists. (D. Savitz, Principal Investigator with doctoral student, E. John), National Institute for Occupational Safety and Health, $30,000, October 1, 1987-May 31, 1990.

Adverse Pregnancy Outcomes among Cosmetologists. (D. Savitz, Principal Investigator with doctoral student, E. John), March of Dimes, $53,000, December 1, 1987-March 31, 1990.

Development of Capabilities for Microcomputer Data Analyses of Epidemiologic Data (D. Savitz, Principal Investigator), University of North Carolina Junior Faculty Development Award, $3,000, January 1, 1988-December 31, 1988.

Epidemiology of Pregnancy Outcome in a Textile Community (D. Savitz, Principal Investigator), National Institutes of Health, National Institute of Child Health and Development, $350,000, March 1, 1988-February 28, 1993.

Menstrual Cycle Patterns and Risk of Breast Cancer (D. Savitz, Principal Investigator with doctoral student, E. Whelan), National Institutes of Health, National Cancer Institute, $25,000, August 1, 1988-December 31, 1989.

The Effect of Exposure to Mercury Vapor and Nitrous Oxide on the Risk of Spontaneous Abortion among Female Dental Assistants (D. Savitz, Principal Investigator with doctoral student, A. Rowland), March of Dimes, $50,000, January 1, 1989-December 31, 1990.

Phenoxy Herbicides and Spontaneous Abortions in Ontario (D. Savitz, Principal Investigator with doctoral student, T. Arbuckle), National Institutes of Health, National Institute of Environmental Health Sciences, $404,982, May 15, 1991 - April 30, 1995.

Lead in Pregnancy, Hypertension, and Neonatal Health (I. Hertz-Picciotto, Principal Investigator, D. Savitz, Co-Investigator), National Institutes of Health, National Institute of Environmental Health Sciences, $855,694, August 1, 1991 - July 31, 1994.

Case-Control Study of Risk Factors in Neuroblastoma (A. Olshan, Principal Investigator, D. Savitz, Co-Investigator), National Institutes of Health, National Cancer Institute, $1,211,736, September 30, 1991 - August 31, 1997.

Genetic susceptibility and dietary factors in ovarian dysfunction: galactose consumption, metabolism, and reproductive impairment (D. Savitz, Principal Investigator with doctoral student, Glinda Cooper), March of Dimes Birth Defects Foundation, $39,216, January 1, 1992 - July 31, 1995.

Mercury and Reproductive Health in Women Dentists (D. Savitz, Principal Investigator, L. Kaste, Co-Investigator), National Institutes of Health, National Institute of Dental Research, $34,245, May 1, 1992 - April 30, 1993.

Parental Occupation and Pregnancy Outcome: Analysis of the National Maternal and Infant Health Survey (D. Savitz, Principal Investigator), March of Dimes Birth Defects Foundation, $54,285, April 1, 1993 - March 31, 1995.

Parents' Drinking, Toxicant Interactions, and Pregnancy (D. Savitz, Principal Investigator), National Institute of Alcoholism and Alcohol Abuse, $50,000, July 1, 1993 - June 30, 1995.

Psychosocial Risks and Preterm in African-American Women, (D. Savitz, Principal Investigator), Centers for Disease Control, $122,222 October 1, 1996 - September 30, 1998.

Cancer Mortality in Minority Workers (D. Loomis, Principal Investigator, D. Savitz, Co-Investigator), National Institute of Occupational and Statistical Health, $243,895, September 30, 1995 - September 29, 1997.

Epidemiology of Preterm Premature Rupture of Membranes (D. Savitz, Principal Investigator), National Institute of Child Health and Human Development, $1,182,246, January 1, 1995 - December 31, 1998.

Pesticides and Breast Cancer in North Carolina (D. Savitz, Principal Investigator), National Institute of Environmental Health Sciences, $883,813, January 1, 1995 - December 31, 1998.

Predictors of Urinary Tract Infection During Pregnancy, (L. Pastore, Principal Investigator, D. Savitz, Co-Investigator), Agency for Health Care and Policy Research, $20,000, September 1, 1995 - December 31, 1996.

Environment and Breast Cancer (D. Savitz, Principal Investigator), National Cancer Institute, $201,793, September 30, 1994 - September 29, 1998.

Supplement to Epidemiology of Preterm Premature Rupture of the Membranes on Cocaine and Preterm Delivery, (D. Savitz, Principal Investigator), National Institute of Child Health and Human Development, $492,914, September 9, 1996 - December 31, 1998.

Research to Advance Environmental Epidemiology: Improving the Use of Human Data in Risk Assessment (D. Savitz, Principal Investigator), US Environmental Protection Agency, $1,584,928, July 1, 1992 - June 30, 1999.

Nutritional Biochemistry and Epidemiology of Cancer Training Grant (L. Kohlmeier, Principal Investigator), National Institute of Environmental Health Sciences, $423,297, July 1, 1997 – June 30, 2002.

Pfiesteria-Related Illness Surveillance and Prevention (C. Moe, Principal Investigator), North Carolina Department of Health and Human Services, $1,362,821, April 1, 1998 – March 31, 2001.

Influence of Iron, Zinc and Folate on Preterm Delivery (D. Savitz, Principal Investigator), National Institute of Child Health and Human Development, $1,405,542, January 1, 1999 – December 31, 2001.

Occupational Exertion and Preterm Delivery (D. Savitz, Principal Investigator), March of Dimes Birth Defects Foundation, $94,877, June 1, 1999 – May 31, 2000.

Psyschosocial Factors in African-American and Preterm Birth, (D. Savitz, Principal Investigator), Association of Schools of Public Health/Centers for Disease Control and Prevention, $170,952, September 9, 1999 – September 8, 2002.

Community-Level Social Influences on Preterm Birth, (D. Savitz, Principal Investigator), Association of Schools of Public Health/Centers for Disease Control and Prevention, $196,791, October 1, 1999 – September 30, 2002.

ANCA Glomerulonephritis: From Molecules to Man (R. Falk, Principal Investigator, D. Savitz, Co-Investigator) National Institute of Diabetes and Digestive and Kidney Diseases $1,070,474, September 1, 2000 – August 31, 2005

Drinking Water Disinfection By-Products and Spontaneous Abortion (D. Savitz, Principal Investigator) American Water Works Association Foundation $3,500,000, November 1, 1999 – September 30, 2005

Reliability, Validity, and Variability in Behavioral Determinants of Drinking Water Disinfection By-Products Exposure (D. Savitz, Principal Investigator) US Environmental Protection Agency $471,000, September 15, 2001 – September 14, 2005

Drinking Water Disinfection By-Products and Male Reproductive Health: Semen Quality and Sperm Biomarkers (A. Olshan, Principal Investigator; D. Savitz, Co-Investigator) US Environmental Protection Agency $557,340, Octdober 1, 2001 – September 30, 2005

Environmental Epidemiology and Exposure Assessment Training Grant Project (L. Kupper, Principal Investigator; D. Savitz, Co-Principal Investigator) National Institute of Environmental Health Sciences $5,642,423, July 1, 2002 – June 30, 2007

Epidemiology of Exertion, Stress, and Preterm Delivery Project (D. Savitz, Principal Investigator) National Institute of Child Health and Human Development $2,154,340, February 1, 2000 – January 31, 2006

Placental Vascular Compromise and Preterm Delivery (J. Thorp, Principal Investigator, D. Savitz, Co-Principal Investigator) National Institute of Child Health and Human Development $2,281,788, September 1, 2001 – August 31, 2006

National Children's Study Duplin County Vanguard Center (B. Entwisle, Principal Investigator; D. Savitz, Original Principal Investigator, consultant). National Institute of Child Health and Human Development $1,091,825, September 30, 2005 – September 29, 2008

Ethnicity and Birth Outcome in New York City (D. Savitz, Principal Investigator) National Institute of Child Health and Human Development, $275,000, January 1, 2006 – December 31, 2008

Risk Factors for Onset and Persistence of TMD (W. Maixner, Principal Investigator; D. Savitz, Co-Investigator through July 2009) National Institute of Dental and Craniofacial Research, $17,216,202, October 1, 2005 – July 31, 2012

National Children's Study Queens County Vanguard Center (P. Landrigan, Principal Investigator; D. Savitz, Co-Investigator). National Institute of Child Health and Human Development $1,091,825, September 30, 2005 – September 29, 2008

C8 and Reproductive and Neurodevelopmental Outcomes (D. Savitz, Principal Investigator, Garden City Group, Inc., $605,693 September 1, 2010 – August 31, 2013

The Epidemiology of Hospitalized Postpartum Depression (D. Savitz, Principal Investigator, NIH 5R21HD0588111-02, $116,629, September 1, 2010 – March 31, 2014

The National Standard for Normal Fetal Growth (E. Chien, Principal Investigator, D. Savitz, Co-Investigator). National Institute of Child Health and Human Development $8,815. September 30, 2010 – December 31, 2011

Metals in Hair and Child Neurobehavioral Development (C. Stein, Principal Investigator; D. Savitz, Subcontract Principal Investigator), Mount Sinai/NIEHS, $41,517, September 1, 2011 – August 31, 2013

Air Pollution and Pregnancy Outcome in New York City (D. Savitz, Principal Investigator), NIH 1-R01 - ES019955, $339,071, April 1, 2011 – March 31, 2015

The epidemiology of postpartum depression and associated childhood outcomes (M. Silverman, Principal Investigator; D. Savitz, Co-Investigator) NIH 1R21HD073010, $24,308, August 1/13- July 31, 2015

Residential Air Pollution and Preeclampsia (D. Savitz, Co-Investigator), NIEHS 1R21ES023073, $204,778, July 1, 2013 – June 30, 2016

Marcellus shale development, respiratory & reproductive outcomes in Pennsylvania (B. Schwartz, Principal Investigator; D. Savitz, Co-Investigator)) NIH 1R21ES023675, $11,748, December 1, 2013 – November 30, 2015

Residential Air Pollution and Preeclampsia (G. Wellenius, Principal Investigator, D. Savitz, Co-Investigator) R21ES023073-01, $160,035, August 9, 2013 – July 31, 2017.

Effect of Iatrogenic Delivery at 34-38 Weeks' Gestation on Pregnancy Outcome (D. Savitz, Principal Investigator) 1R01HD077592-01A1, $345,602, May 15, 2014 – April 30, 2019

CURRENT SUPPORT

Children's Health Exposure Analysis Resource (CHEAR): Coordinating Center (U24) (B. O'Brien, Principal Investigator, D. Savitz, Co-Investigator) 1U24ES026539-01, $2,650,342, September 30, 2015 – August 31, 2019

Environmental Influences on Neurodevelopmental Outcome in Infants Born Very Preterm (B. Lester, Principal Investigator, D. Savitz, Co-Investigator). 5UG3OD023347-02, $3,470,797, September 21, 2016-August 31, 2020
Role: Co-Investigator

PUBLICATIONS:

Published Peer-Reviewed Articles including Research with Original Data, Reviews, and Commentaries

1980

1. Rogers SE, Savitz DA. Toxic substances from coal: Some policy implications for the future. Journal of Environmental Management 1980;11:165-82.

1984

2. Savitz DA, Harley B, Krekel S, Marshall J, Bondy J, Orleans M. Survey of reproductive hazards among Oil, Chemical, and Atomic Workers exposed to halogenated hydrocarbons. American Journal of Industrial Medicine 1984;6:253-64.

3. Savitz DA, Moure R. Review of epidemiologic studies of cancer risk among oil refinery workers. Journal of Occupational Medicine 1984;26:662-70.

1985

4. Savitz DA, Grace C. Determinants of medical record access for an epidemiologic study. American Journal of Public Health 1985;75:1425-6.

5. Savitz DA, Redmond CK. Screening for geographic heterogeneity of disease rates: Application to cancer incidence in Allegheny County, Pennsylvania, 1969-71. Journal of Chronic Diseases 1985;38:145-56.

1986

6. Brinton LA, Huggins GR, Lehman HF, Mallin K, Savitz DA, Trapido E, Rosenthal J, Hoover R. Long-term use of oral contraceptives and risk of invasive cervical cancer. International Journal of Cancer 1986; 38:339-44.

7. Brinton LA, Schairer C, Haenszel W, Stolley P, Lehman HF, Levine R, Savitz DA. Smoking and invasive cervical cancer. Journal of the American Medical Association 1986;255:3265-69.

8. Savitz DA. Changes in Spanish surname cancer rates relative to other Whites in the Denver area. American Journal of Public Health 1986; 76:1210-15.

9. Savitz DA, Hamman RF, Grace C, Stroo K. Respondents' attitudes regarding participation in an epidemiologic study. American Journal of Epidemiology 1986;123:362-6.

1987

10. Alderman BA, Baron AE, Savitz DA. Maternal exposure to neighborhood carbon monoxide and risk of low infant birth weight. Public Health Reports 1987;102:410-4.

11. Brinton LA, Tashima KT, Lehman HF, Levin RS, Mallin, Savitz DA, Stolley PD, Fraumeni JF Jr. Epidemiology of cervical cancer by cell type. Cancer Research 1987;47:1706-11.

12. Savitz DA, Calle EE. Leukemia and occupational exposure to electromagnetic fields. Review of epidemiologic surveys. Journal of Occupational Medicine 1987; 29:47-51.

13. Savitz DA, Zuckerman DL. Childhood cancer in the Denver metropolitan area, 1976-83. Cancer 1987;59:1539-42.

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15. Mayer EJ, Hamman RF, Gay EC, Lezotte DC, Savitz DA, Klingensmith GJ. Reduced risk of insulin dependent diabetes mellitus among breast-fed children. The Colorado IDDM Registry. Diabetes 1988;37:1625-32.

16. Savitz DA. Human studies of human health hazards - comparison of epidemiology and toxicology. Statistical Science 1988;3:306-13.

17. Savitz DA, Pierce NE. Control selection with incomplete case ascertainment. American Journal of Epidemiology 1988;127:1109-17.

18. Savitz DA, Wachtel H, Barnes FA, John EM, Tvrdik JG. Case-control study of childhood cancer and exposure to 60-Hz magnetic fields. American Journal of Epidemiology 1988;128:21-38.

1989

19. Barnes F, Wachtel H, Savitz D, Fuller J. The use of wiring configurations and wire codes for estimating externally-generated electric and magnetic fields. Bioelectromagnetics 1989;10:13-21.

20. Klonoff-Cohen HS, Savitz DA, Cefalo RC, McCann MF. An epidemiologic study of contraception and pre-eclampsia. Journal of the American Medical Association 1989;262:3143-7 (Also published in French, Indian, and Japanese JAMA).

21. Narendrenathan M, Sandler RS, Suchindran CM, Savitz DA. Male infertility in inflammatory bowel disease. Journal of Clinical Gastroenterology 1989;11:403-6.

22. Savitz DA, Baron AE. Estimating and correcting for confounder misclassification. American Journal of Epidemiology 1989;129:1062-71.

23. Savitz DA, Feingold L. Association of childhood cancer with residential traffic density. Scandinavian Journal of Work, Environment and Health 1989;15:360-3.

24. Savitz DA, Pearce NE, Poole C. Methodological issues in the epidemiology of electromagnetic fields and cancer. Epidemiologic Reviews 1989;11:59-78.

25. Savitz DA, Whelan EA, Kleckner RC. Effect of parents' occupational exposures on risk of stillbirth, preterm delivery, and small-for-gestational-age infants. American Journal of Epidemiology 1989;129:1201-18.

26. Savitz DA, Whelan EA, Kleckner RC. Self-reported exposure to pesticides and radiation in relation to pregnancy outcome: Results from the National Natality and Fetal Mortality surveys. Public Health Reports 1989;104:473-7.

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27. Ad Hoc Working Group, International Agency for Research on Cancer. Extremely low-frequency electric and magnetic fields and risk of human cancer. Bioelectromagnetics 1990;11:91-9.

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29. Dole N, Gleiter K, Savitz DA, Chimbira THK, Mbizvo MT. Birth weight patterns in Harare, Zimbabwe. International Journal of Epidemiology 1990;1998-100.

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Baylor College of Medicine
Boston University
Brown University
Dartmouth University
Drexel University
Eastern Virginia Medical School
Emory University
Harvard University
Johns Hopkins University
Memorial Sloan Kettering Cancer Center
Mount Sinai School of Medicine
Michigan State University
New York State Department of Health
Ohio State University
Oregon State University
Robert Wood Johnson Medical School
State University of New York School of Public Health
University of Alabama at Birmingham
University of California, San Francisco
University of Buffalo
University of Chicago
University of Cincinnati
University of Connecticut
University of Michigan
University of Minnesota
University of Pittsburgh
University of Texas
Vanderbilt University

Other Organizations/Research Meetings

American College of Epidemiology
American Conference of Governmental and Industrial Hygienists
Center for Urban Epidemiologic Studies
Health Effects Institute Annual Conference
International Society for Environmental Epidemiology
National Academy of Sciences
National Cancer Center
National Institute of Child Health and Human Development
National Institute of Occupational Safety and Health
Norwegian Epidemiological Society
Population Association of America
Society for Epidemiologic Research SERTalks
Teratology Society

Invited International Presentations

Electromagnetic Fields and Childhood Cancer. Department of Environmental Epidemiology Seminar, Karolinska Institute, Stockholm, 1987

Epidemiologic Studies of Electromagnetic Fields and Cancer. Plenary Presentation, International Society for Environmental Epidemiology, Stockholm, Sweden 1993

Epidemiology of Childhood Cancer, Central Pediatric Hospital, Mexico City, Mexico, 1995

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EXHIBIT B

SAVITZ EXHIBIT B

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